Persistent Postoperative Ventricular Tachycardia Treatment by Using External Cardiopulmonary Support

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Drug-resistant incessant ventricular tachycardia (DRIVT) after cardiac surgery is a rare but almost always fatal event. Antiarrhythmic therapy seems to be ineffective and electrical cardioversion is of limited value when these patients present themselves with recurrent, sustained ventricular tachycardia. A patient with DRIVT in whom external cardiopulmonary support finally succeeded in bringing about resuscitation will be described in this article. The use of external cardiopulmonary support (CPS) should be considered in patients with this kind of malignant arrhythmia.

Introduction

Ventricular arrhythmia is common after cardiac surgery; however, in the majority of cases, postoperative ventricular arrhythmia is easily managed with antiarrhythmic medications. In rare instances, DRIVT occurs after cardiac surgery; however, in most patients, DRIVT results in sudden cardiac death from arrhythmia. External cardiopulmonary support previously has been described as being effective in the treatment of these patients.[1] A patient with postoperative DRIVT in whom external CPS was used successfully to resuscitate him when he went into cardiogenic shock and developed pulmonary edema is reported in this article. The use of CPS allowed the patient to stabilize and permanent effective antiarrhythmic therapy was initiated.

Case presentation

A 71-year-old man was admitted to the hospital with a history of chest pain, orthopnea and paroxysmal nocturnal dyspnea for several months. The electrocardiogram (EKG) revealed sinus rhythm with frequent ventricular premature contractions, QT duration of 0.36 msec, right bundle branch block and left anterior fascicular block. There was no evidence of old or recent myocardial infarction. Acute myocardial infarction was ruled out by testing for normal serial cardiac enzymes and by the absence of new EKG changes.

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Cardiac catheterization was performed and the left ventriculogram revealed anterolateral hypokinesis and an ejection fraction of 40%. Coronary angiography demonstrated a 50% stenosis of the main left coronary artery and occlusion of the distal left circumflex coronary artery.

Surgery was performed with placement of a sequential saphenous venous graft from the aorta to the first and second obtuse marginal coronary arteries and to the first diagnonal branch. A second sequential saphenous venous graft was placed into the left anterior descending and the distal left circumflex arteries.

The patient's immediate postoperative course was uncomplicated; however, on the second postoperative day, following extubation, the patient developed acute respiratory distress. Arterial blood gases on 3 liters per minute of oxygen by nasal cannula showed pO2 of 69.9 mm of Hg and O2 saturation of 94.8%; the chest x-ray revealed a 20% to 30% right apical pneumothorax. A chest tube thoracotomy was performed with subsequent resolution of the pneumothorax. A chest tube thoracotomy was performed with subsequent resolution of the pneumothorax. Frequent ventricular depolarizations were noted on the monitor and the patient then developed sustained ventricular tachycardia.

The patient was cardioverted into sinus rhythm with 200 J and was reintubated; 75 mg of Lidocaine was given intravenously and the maintenance dose was increased from 2 mg per minute to 3 mg per minute. Nevertheless, ventricular tachycardia recurred. The patient was given a total of 1725 mg of Procainamide HCL intravenously, a total of 250 mg of Bretylium Tosylate intravenously, a total of 0.75 mg of Digoxin intravenously and 7 separate cardioversions for ventricular tachycardia and atrial fibrillation with rapid ventricular response of 200. Despite all these measures, ventricular tachycardia persisted and the patient remained hypotensive with systolic blood pressures between 40 and 80; also he was chronically hypoxemic with O2 saturation of 70% on 100% oxygen.

In an attempt to salvage the circulatory collapse and improve oxygenation, external cardiopulmonary support was initiated using percutaneous access through the right femoral artery and vein. Cardiopulmonary support with a blood flow of 3.5 L/min was established with resolution of hypoxemia and hypotension.

Thereafter, amiodarone HCL was given through a nasogastric tube at a dose of 2000 mg a day. The patient improved and CPS

was terminated after 6 hours. No recurrent ventricular tachycardia ensued. The patient was extubated and placed on a regimen of decreasing doses of amiodarone. When he was discharged from the hospital, he had no further ventricular tachycardia.

Six months after surgery, amiodarone was discontinued. Electrophysiologic studies performed at 7 months post-op failed to induce ventricular tachycardia. He was monitored after antiarrhythmics were discontinued, and at 18 months the patient was doing well and was free of clinical arrhythmias.

Discussion

Arrhythmias occur frequently in patients after cardiac surgery.[2] These postoperative arrhythmias range from benign to being potentially life-threatening. Supraventricular arrhythmias are common in the postoperative phase. Atrial fibrillation has been documented to occur in up to 30% of patients who undergo heart surgery and is postulated as being caused by postoperative pericardial inflammation and associated atrial irritability.[2]

Sustained ventricular arrhythmias are much less common. Postoperative ventricular tachycardia has been reported in patients with antecedent histories of ventricular arrhythmia. Ventricular tachycardia in these patients probably is precipitated by postoperative metabolic abnormalities such as hypokalemia and hypomagnesemia, or by excessive circulating catecholamines, changes in ventricular wall stress and hypoxemia. In most patients, postoperative ventricular arrhythmias are not life-threatening, often are self-limiting, and usually are responsive to antiarrhythmic treatment. DRIVT is rare, almost uniformly fatal and the development of alternate therapies for this condition is crucial.[2]

We have described a patient in whom external CPS was used to accomplish successful resuscitation, although the mechanism by which CPS did this is conjectural. We postulate that it resulted in improved oxygenation, improved myocardial perfusion and hemodynamic stabilization; therefore, that possibly decreased catecholamine stress and allowed suppression of incessant ventricular tachycardia that had previously not responded to antiarrhythmic therapy. Incessant ventricular tachycardia and associated pulmonary edema had prevented adequate oxygenation. Cardioversion and ventricular pacing were of limited value in this patient despite temporary successful cardioversion.

Transient control of the arrhythmia is essential because the incessant ventricular arrhythmia could be only temporary. Arrhythmogenesis in the postoperative patient might be the result of a variety of insults that will be self-limiting. The fact that we were able to withdraw our patient from antiarrhythmic therapy 6 months after surgery supports this concept.

Experiences with the use of external cardiopulmonary support in patients with postoperative ventricular tachycardia have been limited. O'Neill and colleagues have described a patient in whom CPS resulted in termination of incessant ventricular tachycardia.[1]

The authors postulated that CPS allowed for a decrease in left ventricular wall tension that resulted in a decrease in ventricular arrhythmogenesis. This concept is supported by San Fellipo[3] who described effective termination of intractable ventricular tachycardia by using an intra-aortic counterpulsation catheter. Irrespective of the mechanism of action, the recognition of potential for the successful treatment of incessant ventricular tachycardia with external cardiopulmonary support is crucial.

References

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